in their cohort as greater urinary levels of vitamin D metabolites have been observed in patients with abnormal urinary protein excretion.[4] Of note, Ishimura *et al.*[5] reported, in 76 Japanese patients with CKD stage III and IV, that diabetic patients had lower 25OHD levels than non-diabetic ($11.4 \pm 5.6\ ng\ ml^{-1}$ vs $22.3 \pm 9.4\ ng\ ml^{-1}$; $P < 0.0001$). Larger representative studies are necessary to evaluate the prevalence of 25OHD deficiency across different ethnics groups and levels of kidney dysfunction.

1. Kosmadakis G, Duja S, Basta M *et al.* 25(OH) vitamin D deficiency amongst SE Asians and Caucasians with CKD 3 and 4, and its role in hyperparathyroidism. *Kidney Int* 2007; **73**: 360.
2. Chonchol M, Scragg R. 25-Hydroxyvitamin D, insulin resistance, and kidney function in the Third National Health and Nutrition Examination Survey. *Kidney Int* 2007; **71**: 134–139.
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Response to '25(OH) vitamin D deficiency among SE Asians and Caucasians with CKD 3 and 4, and its role in hyperparathyroidism'

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Kosmadakis and co-workers have submitted a letter to *Kidney International* that describes the results of a prevalent cohort of 113 South East Asians and Caucasians with chronic kidney disease 3 and 4. They describe in that cohort a deficiency of 25 Vitamin D that is associated with hyperparathyroidism.[1] As these data are essentially presented in abstract form, as a letter, it is difficult to comment in detail.

The issue of ethnicity and Vitamin D levels is important given the differential cardiovascular and bone disease risks, which have been described in chronic kidney disease in different ethnic groups. This short letter to the editor does highlight the issue of ethnicity for the readership. The SEEK paper[2] reported results on a large cohort, which included a substantial cohort of African Americans. Differences in that cohort were also described, but were not the major focus of the paper.

Of note, two manuscripts have been submitted, one from the SEEK (US) database and one from a different Canadian provincial database, which further address the issue of ethnicity and differences in prevalence of abnormalities of mineral metabolism. Together, these reports, including the letter from these authors, suggest that the international community need to review data in the context of ethnicity so as to ensure robust conclusions and appropriate design of therapeutic trials.

Larger studies are needed to investigate these associations and their implications in chronic kidney disease patients. The data to data differences are interesting and suggestive, but a substantial amount of further research, in both epidemiology and pathophysiology, is needed before one advocates for large-scale therapeutic interventional trials based on observations of single deficiencies.

1. Kosmadakis G, Duja S, Basta M *et al.* 25(OH) Vitamin D deficiency among SE Asians and Caucasians with CKD 3 and 4, and its role in hyperparathyroidism. *Kidney Int* 2007; **73**: 360.
2. Levin A, Bakris GL, Molitch M *et al.* Prevalence of abnormal serum vitamin D PTH, calcium, and phosphorous in patients with chronic kidney disease: results of a study to evaluate early kidney disease. *Kidney Int* 2007; **71**: 31–38.

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Glucose transport across the proximal tubule brush border membrane: Response to diabetes mellitus

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To the Editor: Lee *et al.*[1] recently addressed the implications for diabetic tubulopathy of reduced sodium-dependent glucose transport across the proximal tubule brush border membrane (BBM) during hyperglycemia. We believe that the discussion gives an incomplete view of how BBM glucose transport is influenced by diabetes mellitus.

Our own studies have failed to detect changes in sodium-dependent glucose transport-mediated glucose uptake across the BBM during experimental diabetes mellitus.[2] Interestingly, however, we found that diabetes promoted facilitated (facilitated glucose transports (GLUT)-mediated) glucose uptake across this membrane,[2] this being largely due to an eightfold increased BBM expression of GLUT2. Normalization of plasma glucose reduced GLUT-mediated uptake and GLUT2 expression to non-diabetic levels. Additional work[3] implies that increased BBM levels of protein kinase C-β1 isoform may be involved in the diabetes-induced appearance of GLUT2 at the BBM. Indeed, we found strong positive linear correlations between plasma glucose level (5–40 mmol/l) and BBM expression of both GLUT2 and protein kinase C-β1 and between GLUT2 and protein kinase C-β1.